

--Assay of the truncation mutants proved to be a sensitive and specific screen for the identification of the MAD2/ER beta interaction domain. The interaction domain was identified as encompassing nucleotides 516 to 622 of ER beta (Fig. 3A). Fig. 3B summarizes the two hybrid protein interaction results. As is shown in Fig. 3B, the ER beta/MAD2 interaction domain is defined by nucleotides 516 to 641 of ER beta that interact with MAD2 clone EC1. Fig. 3B also shows that slightly larger regions containing the interaction domain support the interaction between ER beta and MAD2, while fragments lacking nucleotides 516-622 of ER beta do not.--

Please amend the specification on page 20, lines 3-15, to read as follows.

--Thus the GST-fusion protein experiments demonstrate that mER $\beta$  is brought down, or associates with, the GST-MAD2 clone and, in the converse experiment, MAD2 is brought down by GST-mER $\beta$ . Each case demonstrates the protein-protein interaction. In contrast, the results shown in Fig. 4C indicate that while GST-mER $\beta$ , as expected, brings down ER $\alpha$  (this is a positive control since it is known that these two proteins heterodimerize), GST alone, or GST MAD2, shown in the third and fourth lanes, respectively, do not bring down ER $\alpha$ . This result confirms the two hybrid data, i.e. that ER $\alpha$  does not interact with MAD2. Fig. 4D, which shows the results of protein- protein interaction studies between MAD2 and ER beta mutants, also confirms the two hybrid data which identified the MAD2/ER beta interaction domain as including nucleotides